

METABOLIC BONE DISEASES...

K.M

د. خالد حيلدار

metabolic bone diseases are :-

- hyperparathyroidism
- nutritional rickets
- nutritional osteomalacia.
- infantile scurvy.

(I) HYPERPARATHYROIDISM

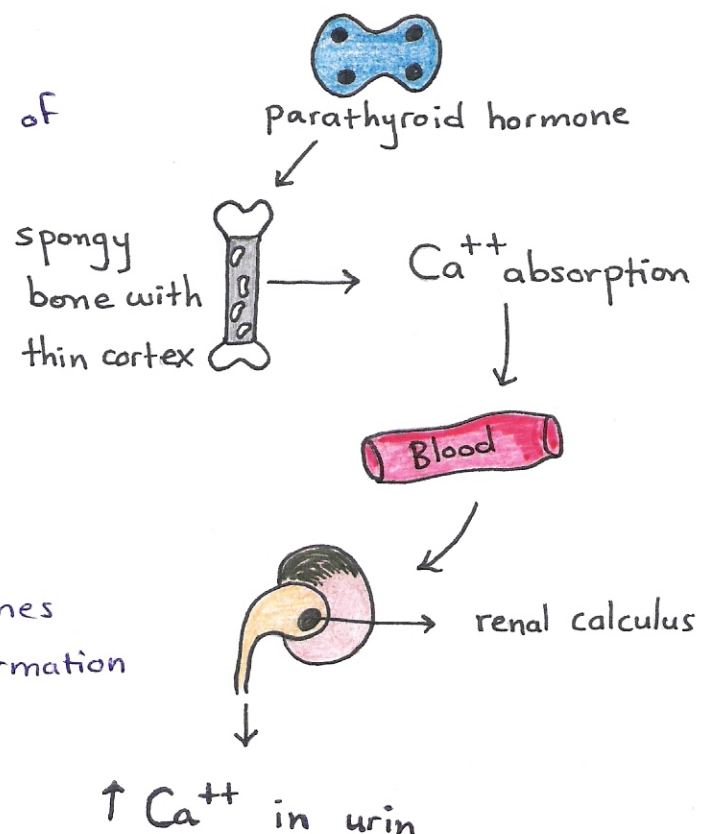
hyperparathyroidism is characterized by :-
lassitude (weakness) , dyspepsia , generalized osteoporosis
& cystic changes in some bones.

Cause ↗

it's caused by excessive secretion of parathyroid hormones usually from adenoma of one of parathyroid glands.

Pathology ↗

- excessive secretion of PTH lead to generalized absorption of Ca^{++} from the bone
- the Ca^{++} is liberated into the blood & excreted in large quantities in the urin
- the bone become spongy & the cortex become thin
- cystic changes often develop in one or more of long bones
- the risk of renal calculi formation is increased.



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Clinical picture →

the Pt is adult, having :-

- bone pain
- weakness & indigestion.
- there might be deformity from bending of softened bone
- there might be deformity from pathological *

Radiological Features →

- rarefaction of the whole skeleton (the loss of density might be very marked.
- cortex is very thin
- scattered cystic changes may present in long bones
- radiograph of renal tract may show stones.

Investigations →

- plasma Ca^{++} is ↑↑
- plasma inorganic Ph is ↓
- ↑ Ca^{++} & Ph excretion in urin.

treatment →

the causative parathyroid tumor should be located & removed.

(II)

NUTRITIONAL RICKETS

it's defective calcification of growing bone in consequence of distributed Ca^{++} & ph metabolism.

Cause →

it's caused by deficiency of vit D in diet & inadequate exposure to sunlight which promote synthesis of vit D in the body.

Pathology →

- vit D promote the absorption of Ca^{++} & ph from intestine
- therefore deficiency of vit D lead to inadequate absorption of Ca^{++} & ph.
- so the level of Ca^{++} in the blood can then be maintained only by skeletal Ca^{++} .
- this will lead to proliferating osteoid tissue in the growing epiphysis thus remains uncalcified
- this will lead to generalized softening of bones.

Clinical Features →

usually occur in children aged one year
the predominant signs are :-

- large head
- retarded skeletal growth
- enlarged epiphysis
- curving of long bones
- costochondral junction enlargement [Ricky Rosony]
- [Harrison's sulcus] develop where the diaphragmatic attachments pull on softened ribs.

Radiological features ~

- generalized loss of density of the skeleton with cortex softening.
- the most striking changes are in growing epiphysis which are :-
 - broad vertical epiphyseal lines ↑
 - widening of epiphysis laterally
- end of the shaft become cupped & brush.
- the bending of bones might be obvious.

Investigations ~

- plasma Ph level is ↓ while Ca^{++} is NORMAL
- ALP is ↑ ~ it's an indicator for severity of the disease & for response to ttt
- measurement of serum level of 25 hydroxy vit D is helpful.

Diagnosis ~

AP radiograph of wrist joint should be taken w̄ will show diagnostic changes.

treatment ~

- it usually responds well to vit D & sun exposure
- severe bone deformity should be corrected by osteotomy or osteoclasia.

Other forms of rickets :-

the characteristic epi. changes seen in rickets occur in other Ds in w̄ any factor responsible for disordered Ca^{++} & Ph is differ & there are 4 types :-

- 1- familial hypophosphatemia
- 2- Cystinosis
- 3- uremic osteodystrophy
- 4- Coeliac (gluten induced rickets)

(III)

NUTRITIONAL OSTEOMALASIA

it's the adult counterpart of infantile nutritional rickets.

Cause & pathology ↪

- as in infantile rickets there is deficiency of vit D « & often of Ca^{++} » in diet.
- the bone matrix remain but it's mineralization is deficient.
- the bone trabeculae are not abnormally thin but they are largely composed of poorly calcified osteoid tissue.
- there is abundance of fibrous tissue in the marrow.

Clinical Feature ↪

- the main features are : bone pain & deformity.
- bone ~~is~~ is common.

Radiographic changes ↪

- rarefaction of the whole skeleton.
- bone cortex is abnormally thin.
- the long bones may be curved.
- multiple spontaneous fractures

Characteristic

- [LOOSER'S ZONES] represent stress ~~is~~ which heal & fail to mineralize, these zones may be shown in the ribs, pelvic rim ---

N.B ↪ osteomalasia **CAN'T** be differentiated from rickets by x-ray as in both states the bone looks thin & rarefied

Investigations ~

- plasma Ca^{++} is NORMAL or LOW.
- plasma Ph is ↓
- ALP ↑
- serum level of 25 hydroxy vit D is ↓

treatment ~

- adequate diet & vit D & Ca^{++} supplements.
- osteotomy may be required to correct deformity.

Other forms of osteomalacia :-

in adults, osteomalacia may develop from causes other than purely nutritional, such causes include :-

- 1- advanced renal disease (e.g CRF)
- 2- malabsorption syndrome as ~
 - chronic obstruction of bile duct
 - chronic pancreatic diseases
 - idiopathic steatorrhea.

N.B

BONE DISEASE	Ca^{++}	phosphate	ALP	PTH	NOTES
osteoporosis	N	N	N	N	↓ bone mass
osteopetrosis	N	N	N	N	thick dense bone [marble bone]
osteomalacia & Rickets	↓	↓	variable	↑	soft bones
osteitis fibrosa cystica	↑	↓	↑	↑	Brown tumor (hyperparathyroidism)
Paget's disease of bone	N	N	↑	N	abnormal architecture (loss of demarcation)

(IV)

INFANTILE SCURVY

it's a haemorrhagic disease caused by vitC deficiency in diet

Pathology ↪

- most of changes are in long bones in which there is a lack of **OSTEOBLASTIC** activity in **EPIPHYSEAL PLATE**
- the Hge (bleeding) may occur from :-
 - epiphyseal plate
 - gums
 - within the orbit
- the onset is rapid with loss of use of the limb b/c of pain [**pseudoparalysis**]
- the limb is swollen & tender

Radiographic features ↪

- dense line betn metaphysis & epiphyseal cartilage
- clear band of rarefaction on diaphyseal side of epiphyseal cartilage.
- ossification in subperiosteal haematoma & as a result the bone will become thickened.

N.B ↪ the clear zone in metaphysis indicate the arrest of osteoblastic activity.

